CASE REPORT

Transient left ventricular systolic dysfunction mimicking myocardial infarction after pericardiocentesis

R. W. J. H. Weijers · J. C. Post

Published online: 8 May 2013 © The Author(s) 2013. This article is published with open access at Springerlink.com

Case

A 69-year-old woman was admitted to our hospital because of weight loss and progressively worsening dyspnoea. Her previous medical history was unremarkable. She was admitted to the internal medicine department. A cardiologist was consulted because of atrial fibrillation with rapid ventricular response and an enlarged cardiac silhouette on her chest Xray. On physical examination her blood pressure was 120/70 mmHg with a heart rate of 120 beats/min. She had engorged jugular veins and an enlarged liver without peripheral oedema. Her ECG showed atrial fibrillation and low voltage. No Q waves were noted. An echocardiogram showed a large pericardial effusion of more than 30 mm and a swinging heart (Fig. 1). The left ventricle was small and hyperdynamic without wall motion abnormalities. There was no collapse of the right ventricle. The inferior vena cava was dilated without inspirational collapse. Because of imminent cardiac tamponade a pericardiocentesis was performed with the immediate evacuation of 800 cc of haemorrhagic pericardial fluid. Six hours after this procedure control echocardiography was performed which showed only a small amount of pericardial fluid, but now a poor left ventricular function with general hypokinesia and anterior and septal akinesia. T-wave inversion and Q waves were seen on the ECG in the anterolateral leads (Fig. 2) but the cardiac markers were not elevated. She was treated with ACE inhibitors, diuretics and low-dose beta blockade. Analysis of the pericardial fluid showed malignant cells and on a chest CT scan a mass was detected. Subsequently, she was diagnosed with small cell lung carcinoma (SCLC) stage IV

R. W. J. H. Weijers (🖂) · J. C. Post

Department of cardiology, Catharina Hospital Eindhoven, PO Box 1350, 5602 ZA Eindhoven, the Netherlands e-mail: ralphweijers@online.nl with metastasis to the pelvis, pericardium and brain and treated with radiation therapy. She was discharged and returned several months later with complaints of fatigue and progressive dyspnoea. Echocardiography again showed a large pericardial effusion; the left ventricle now had a normal systolic function without wall motion abnormalities. The ECG now showed normal R progression in the anterior leads with disappearance of Q waves (Fig. 3). She was again treated with pericardiocentesis and was discharged the following day. After this episode she died at home as a consequence of her progressive lung carcinoma.

Discussion

Pericardiocentesis for cardiac tamponade is a commonly performed procedure which may lead to complications such as perforation of the (right) ventricle, laceration of a coronary artery, pneumothorax and ultimately death. Transient left ventricular dysfunction has been described by various authors [1-7]. Case reports describe patients with pulmonary oedema [1, 3, 5], cardiogenic shock [2, 4] and acute adult respiratory distress syndrome [6]. Global myocardial dysfunction [4, 5] as well as regional dysfunction [1, 2], as in our case, are described. The underlying mechanism is poorly understood. Multiple factors are considered to play a role. During tamponade, external compression of the myocardium leads to reduced stroke volume and cardiac output. Compensatory mechanisms include tachycardia and expansion of the intravascular volume. Catecholamine levels are generally high with vasoconstriction [8]. Sudden release of pericardial constraint through pericardiocentesis could lead to a disproportionate increase in end-diastolic volume of the right ventricle compared with the left ventricle and a temporary mismatch in ventricular outputs. This could lead to a sudden increase in left ventricular end-diastolic pressure and, in the presence of high



Fig. 1 Initial echocardiogram showing a large pericardial effusion and a hyperdynamic left ventricle with normal wall motion and thickness

afterload through vasoconstriction and a suddenly developed normal or negative intrapericardial pressure, to an acute increase in left ventricular wall stress and left ventricular failure [2]. Other mechanisms postulated are myocardial stunning because of mismatch of oxygen distribution across the myocardial wall or altered coronary blood flow due to high pericardial pressures [7]. In our case there was akinesia of the anterior wall and developing Q waves in the anterior leads, but no elevation of the cardiac enzymes, excluding myocardial infarction. Also, the regional akinesia as well as the Q waves and negative T waves appeared to be completely reversible. Other authors have performed myocardial perfusion studies [1] or coronary angiograms [2] in cases like this but found no evidence of coronary artery disease. The exact mechanism for this observation is not known. Other reversible cardiomyopathies, such as thyrotoxicosis-induced cardiomyopathy, Tako Tsubo cardiomyopathy or peripartum cardiomyopathy are also known to produce these ECG changes and echocardiographic abnormalities [9, 10]. Because of the possible impact on the haemodynamics of the acute left ventricular overload which can follow abrupt relief of cardiac tamponade, it might be wise not to remove a large pericardial effusion too quickly. In cardiac tamponade intrapericardial pressure declines rapidly after evacuation of the first 50-200 ml of pericardial fluid. Therefore some authors have suggested that pericardial fluid should be removed only until tamponade physiology disappears and after that gradually [1-3].

Conclusion

Reversible left ventricular dysfunction is a rare complication of the abrupt evacuation of pericardial fluid in the setting of a pericardial tamponade. Regional akinesia and developing Q waves resembling anterior infarction are also possible, as shown in our case, but the mechanisms of this myocardial pseudo-infarction and transient left ventricular dysfunction are not completely understood.



Fig. 2 The ECG on admission (top) showing atrial fibrillation and low voltage and after pericardiocentesis (bottom) showing Q waves and T-wave inversion in the anterolateral leads



Fig. 3 The ECG on re-admission showing normal R-wave progression and disappearance of Q waves

Conflict of interest None declared.

Open Access This article is distributed under the terms of the Creative Commons Attribution License which permits any use, distribution, and reproduction in any medium, provided the original author(s) and the source are credited.

References

- Bernal JM, Afonso L, Pradhan J, et al. Acute pulmonary edema following pericardiocentesis for cardiac tamponade. Can J Cardiol. 2007;23:1155–6.
- Chamoun A, Cenz R, Mager A. Acute left ventricular failure after large volume pericardiocentesis. Clin Cardiol. 2003;26:588–90.
- Vandyke WH, Cure J, Chakko CS, et al. Pulmonary edema after pericardiocentesis for cardiac tamponade. N Engl J Med. 1983; 309:595–6.

- 4. Braverman AC, Sundaresan S. Cardiac tamponade and severe left ventricular dysfunction. Ann Intern Med. 1994;120:442.
- 5. Wolfe MW, Edelman ER. Transient systolic dysfunction after relief of cardiac tamponade. Ann Intern Med. 1993;119: 42-4.
- Glasser F, Fein AM, Feinsilver SH, et al. Non-cardiogenic pulmonary edema after pericardial drainage for cardiac tamponade. Chest. 1988;94:869–70.
- 7. Yoshiro H, Shuji, Norio U, et al. Severe circulatory collapse immediately after pericardiocentesis in a patient with a chronic cardiac tamponade. Anesth Analg. 1993;77:1278–81.
- Spodick DH. The normal and diseased pericardium: current concepts of pericardial physiology. J Am Coll Cardiol. 1983;1:240–51.
- 9. Bird-Lake E. Severe transient left ventricular dysfunction induced by thyrotoxicosis. Neth Heart J. 2011;19:352–4.
- Lok SI, Kirkels JH, Klöpping C, et al. Peripartum cardiomyopathy: the need for a national database. Neth Heart J. 2011; 19:126–33.